geted for PV isolation. Circumferential PV electrograms around the PV ostium were acquired simultaneously using a circular catheter and used to guide ablation at ostial sites with the earliest PV potentials during sinus rhythm or coronary sinus pacing. Radiofrequency ablation was performed at 50 C and 20-30 W. The endpoint was the elimination of either AF (42 patients) or atrio-PV conduction based on the abolition of distal PV potentials (38 patients).
Results: Focal ablation, PV isolation or both were performed in 42, 19 and 19 patients, respectively. Of the 104 foci identified, 94 triggers (90%) originated from the PV single in 47% and multiple in 53% and 10 originated from the atrial tissue. AF recurred in 42 patients and re-ablation was performed in 24: 7 from the same source, 3 from non-PV foci, 2 from a different part of the same PV, 2 from a different PV, 2 from PV ostia proximal to the previous PV isolation, and 8 due to recovery of atrio-PV conduction. During a mean follow-up of 14 ± 9 months, the clinical success rate with combined therapy (89%) was significantly higher than those with focal ablation (57%) or PV isolation (53%) without drugs ($p < 0.05$). No PV stenosis was observed.
Conclusions: Focal ablation did not prevent AF by another focus in the same PV or other PVs. The success rate of PV isolation is relatively low, probably due to unmasked foci from the PV ostial edge or atrial tissue. Combined treatment with focal ablation and PV isolation is more effective for treating AF than either approach alone.

1208-116 Pulmonary Vein Isolation During Minimally Invasive Mitral Valve Surgery: One-Year Follow-Up

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Background: Atrial fibrillation (AF) complicating mitral valve (MV) disease often fails to respond to medical management. Surgical and catheter based procedures that electrically isolate atrial regions have been successful at restoring sinus rhythm (SR). We report our one-year follow-up for patients undergoing pulmonary vein (PV) isolation during minimally invasive MV surgery.
Methods: Twenty-two patients (11 men and 11 women; age 59±6 years) underwent mitral valve surgery and pulmonary vein isolation. Sixteen had chronic AF (2years±8months) and 6 had paroxysmal AF (PAF). Left atrial size was 5.6±1.2 and 4.9±0.6 cm for chronic AF and PAF patients respectively. MV repair was performed with a minimally invasive port access approach in 15 patients, 7 required sternotomy. A flexible, 8-coil radiofrequency ablation probe was used to isolate the pulmonary veins. Both right and left pulmonary veins were independently encircled. Lesions were also extended between the PV islands, the oversewn left atrial appendage, and the MV annulus. Additional procedure time was 11±2 min. Patients were cardioverted to SR intraoperatively.
Results: At discharge, 11 of 16 chronic AF patients were in SR. With antiarrhythmic drugs or cardioversion, 4 additional patients returned to SR. 3 other patients developed atrial flutter; 2 returned to SR with antiarrhythmic drugs, one degenerated to AF after multiple attempts at ablation of atypical flutter. At follow-up (17±5 months) patients were evaluated for atrial rhythm, contraction, and size. 13 of 16 chronic AF patients remained in SR; one patient had left atrial standstill, the others had normal biatrial function. All PAF patients maintained SR and normal atrial contraction. 37% of the cohort required antiarrhythmic drugs or cardioversion, generally within the first 3 months after surgery. Follow up sinus rhythm was associated with a significant reduction of left atrial size (5.6 to 4.7 cm, p<0 .001).
Conclusion: Radiofrequency pulmonary vein isolation performed during minimally invasive and standard mitral valve repair was successful at restoring SR to 81% of chronic AF patients. To achieve these results, however, attentive medical management is necessary.

POSTER SESSION

1209 Defibrillation: Basic Science

Tuesday, March 19, 2002, 3:00 p.m.-5:00 p.m.
Georgia World Congress Center, Hall G
Presentation Hour: 4:00 p.m.-5:00 p.m.

1209-106 Test of a New Reduced-Current Biphasic Waveform in Transthoracic Defibrillation of Canines

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Background - Biphasic truncated exponential (BTE) waveforms are used in all implantable cardioverter defibrillators, and are now being implemented in external transthoracic defibrillators. A new BTE waveform defibrillator has been developed (Medical Research Labs (MRL), Inc; Buffalo Grove, IL) that can deliver substantially lower peak currents at equal the delivered energy than currently available BTE waveform defibrillators. We compared the transthoracic defibrillation efficacy of the MRL BTE waveform to the standard Edmark waveform.
Methods - Canines (n=5, 71±7 lbs) were anesthetized with 20 mpk sodium pentothal I.V., and maintained with additional infusions as required. Fibrillation was induced with 60 Hz current via a bipolar pacing catheter in the right ventricle. The chest was shaved and defibrillating patch electrodes (R2, 3200-1715) were placed on the left and right chest walls. Defibrillation energy required for 50 percent success probability (ED50) was estimated by using an up-down protocol. ED50 estimations were run in parallel, in each case starting with a shock strength of 50 Joules. The waveforms being compared were alternated every other fibrillation induction. Termination of fibrillation with return of spontaneous circulation was achieved in each instance either by the selected energy or with a rescue shock, and the next episode followed after a 4- minute recovery. ED50 peak current and energy were estimated for each animal by logistic regression analysis.
Results - This study consisted of 82 total fibrillation/defibrillation episodes, and the mean impedance for these animals was 62 ± 8.8 ohms. Mean ED50 delivered energy for the Edmark waveform was 35.3 J and for the BTE waveform was 26.3 J ($p = 0.014$). Mean ED50 peak current for the Edmark waveform was 16.6 A, and for the BTE waveform was 6.4 A ($p<0.001$).
Conclusion - The new BTE waveform was more effective than the Edmark waveform, requiring 25% less delivered energy, and 61% less peak current in this canine model.

1209-107 Intravenous Bolus Amiodarone Administration Exhibits an Immediate and Gradual Increase in Ventricular Fibrillation Threshold: Experimental Study

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Backround: The aim of the study was to examine the early time course of changes in ventricular fibrillation (VFT) and defibrillation (DFT) thresholds after an i.v. bolus of amiodarone (A) in an experimental pig model of transient myocardial ischemia.
Methods: VFT and relative effective ventricular refractory period (ERP) were measured in 15 anesthetized open-chest pigs after 3min of regional coronary ischemia before (time 0) and 2, 15, 30, 60 and 90 min after a 5 mg/kg i.v. bolus of A injected within 15 sec (Group I, n=10) or normal saline (Group II, n=5). DFT was also measured by systematically increasing the stored voltage until defibrillation was accomplished. Hemodynamics, acid-base balance and temperature were kept stable throughout the experiments.
Results: The time course of VFT, ERP and DFT in the 2 study groups was as follows:(see table)
Conclusion: Intravenous bolus administration of amiodarone increased VFT and ERP steadily over time, reaching a plateau 60 minutes after its administration, without any effect on DFT.

	Time (min)	0	2	15	30	60	90
Control	VFT (mA)	9.5±6.7	11.4±9.8	9.6±6.1	9.4±5.3	8.2±6.7	9.4±4.2
Amiodarone (5mg/kg)	VFT (mA)	9.2±4.6	11.4±8.4	13.7±6.5*#	34.2±28.7*#	50.3±37.8*#	53.2±38.8*#
Control	DFT (J)	11±5.7	13±6.7	10.6±6.1	12±9.2	7.2±7.2	11.8±12.5
Amiodarone (5mg/kg)	DFT (J)	14.5±15.2	14.2±14.8	9.3±9.4	12.2±7.3	12±14.5	7.2±3.1
Control	ERP (msec)	205±32	206±30	212±33	205±31	186±31	178±28*
Amiodarone (5mg/kg)	ERP (msec)	197±26	204±25	211±38	212±40*	220±34*#	227±32*#

*p<0.05 vs Time 0
#p<0.05 vs Control

1209-108 Kinetics of Shock Induced Transmembrane Polarization: Effect of Ischemia

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The time constant (τ) has been used to quantify the cellular response to a defibrillation shock. However, τ has been previously characterized in normal healthy hearts, rarely the case in the clinical setting. The purpose of this study was to measure τ during ischemia. **Methods:** Optical techniques mapped anterior, epicardial, electrical activity during defibrillation shocks free of shock-induced artifacts. Monophasic shocks (100, 160, 220 V; 150 μF; 8 ms) of either polarity were applied at 50% of the action potential duration using a transvenous lead system analogous to a hot-can configuration in 5 Langendorff-perfused rabbit hearts. Global ischemia (I) was introduced by reducing the perfusion flow to ¼ of that used during control (C), and the protocol was repeated. The polarizations were approximated with single-exponential fits from a total of 6805 recordings using the Levenberg-Marquardt method. **Results:** See graph. The positive τ increases with ischemia were statistically significant ($p<0.01$) for the +160 and +200 V shocks, magnitudes at and above the defibrillation threshold. The negative τ differences were not significant. **Conclusion:** Global ischemia has a small but sometimes significant effect on the cellular response to a defibrillation shock. We speculate that the positive τ increases with ischemia are related to an increase in the intracellular resistance caused by gap junction uncoupling. However, the differences in the effect of ischemia on positive and negative shock τ warrant further study.